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# Morbilliviral infections in marine mammals[1]

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ABSTRACT

Epizootics of infectious disease were unknown in cetaceans prior to 1987. However, since then there have been at least three epizootics in dolphins and two in pinniped species. Many of the clinical, pathological and epidemiological features of these events were similar to those of morbilliviral infections in terrestrial mammals. There has been speculation that contaminants may have predisposed marine mammals to these and this is discussed. Morbilliviruses are highly pathogenic viruses and caused epizootics in terrestrial mammals long before the advent of anthropogenic contaminants.

KEYWORDS: POLLUTION; DISEASE; EPIZOOTICS; IMMUNOSUPPRESSION; PATHOLOGY; PINNIPEDS; STRIPED DOLPHIN; HARBOUR PORPOISE; BOTTLENOSE DOLPHIN; WHITE WHALE; REVIEW

## REPORTED INFECTIONS

Recognised morbillivirus infections had not been reported in aquatic mammals prior to 1987, but since then there have been a number of epizootics of morbilliviral disease in cetacean and pinniped populations in several regions of the world (e.g. Simmonds, 1992).

### Pinnipeds

An epizootic of morbillivirus infection killed approximately 18,000 harbour seals (*Phoca vitulina*) and several hundred grey seals (*Halichoerus grypus*) in Europe in 1988 (Kennedy *et al.*, 1988b; Osterhaus and Vedder, 1988; Dietz *et al.*, 1989; Bergman *et al.*, 1990; Kennedy, 1990). This die-off apparently began along the Baltic coast in April of that year and subsequently spread to seal colonies along the North Sea coasts of Norway, Sweden, Denmark, Germany, The Netherlands, the United Kingdom and Ireland. It appears to have terminated in late 1988 although a small localised outbreak occurred in northern Norway in late 1989 (Krogsrud *et al.*, 1990). In addition, a morbilliviral epizootic killed several thousand Baikal seals (*Phoca sibirica*) in Lake Baikal from late 1987 to late 1988 (Grachev *et al.*, 1989).

Laboratory studies (Cosby *et al.*, 1988; Curran *et al.*, 1990; Blixenkrone-Moller *et al.*, 1992; Rima *et al.*, 1992) indicated that the morbillivirus which infected European seals was a newly recognised virus (the phocine distemper virus, PDV). Similar studies of a virus isolated from Lake Baikal seals (Osterhaus *et al.*, 1989; Visser *et al.*, 1990; Barrett *et al.*, 1992) indicated that it was a strain of canine distemper virus (CDV). No epidemiological link could therefore be established between the Siberian and European epizootics.

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**Cetaceans**

The first evidence of morbillivirus infection in cetaceans emerged from the coast of Ireland during the 1988 European seal epizootic. At that time a morbillivirus disease was diagnosed in six harbour porpoises (*Phocoena phocoena*) found stranded on the coast of Northern Ireland (Kennedy *et al.*, 1988a; 1991). Morbilliviral disease was subsequently found in a few harbour porpoises found stranded on the coasts of England, Scotland and The Netherlands in 1990 (Kennedy *et al.*, 1992; Visser *et al.*, 1993). As the porpoises from the coast of Northern Ireland were found in a region inhabited by morbillivirus-infected harbour seals, it was originally believed that interspecific transmission of PDV had occurred. However, the porpoise morbillivirus was subsequently isolated and characterised as another newly discovered morbillivirus distinct from PDV (McCullough *et al.*, 1991; Welsh *et al.*, 1992; Barrett *et al.*, 1993; Visser *et al.*, 1993; Blixenkrone-Moller *et al.*, 1994).

A die-off of striped dolphins (*Stenella coeruleolba*) began along the Mediterranean coast of Spain in July 1990 (Domingo *et al.*, 1990; 1992; Duignan *et al.*, 1992). This epizootic rapidly spread to other areas of the Spanish and French Mediterranean coasts and probably also to the coasts of Morocco and Algeria. It subsided in late 1990 but re-emerged along the southern coast of Spain between June and September 1991 and eventually reached the southern Adriatic Sea, Ionian Sea, Sicilian Channel and southern Tyrrhenian Sea. A third outbreak occurred in the region of the Greek Islands in early 1992. At least several thousand animals are believed to have died during these Mediterranean outbreaks (Di Guardo *et al.*, 1992; 1995; Aguilar and Raga, 1993). Laboratory characterisation of a morbillivirus isolated from affected dolphins in the western, central and eastern regions of the Mediterranean Sea indicated that all three outbreaks were phases of a single epizootic (Van Bressem *et al.*, 1993).

It has been suggested that the northwestern European harbour porpoise and Mediterranean striped dolphin viruses may represent two distinct strains of the same morbillivirus (Bolt and Blixenkrone-Moller, 1994). Antigenic and genomic analyses indicate that the viruses isolated from these cetaceans are distinct from other morbilliviruses including the newly recognised PDV (Curran *et al.*, 1990; 1992; McCullough *et al.*, 1991; Welsh *et al.*, 1992; Barrett *et al.*, 1993; Van Bressem *et al.*, 1993; Visser *et al.*, 1993; Blixenkrone-Moller *et al.*, 1994; Bolt and Blixenkrone-Moller, 1994). Although they cause a distemper-like disease in harbour porpoises and striped dolphins (Kennedy *et al.*, 1988a; 1991; 1992; Domingo *et al.*, 1992; Duignan *et al.*, 1992), the cetacean morbilliviruses appear to be more closely related to rinderpest, peste-des-petits-ruminants and measles viruses than to CDV and PDV, which comprise the distemper subgroup of morbilliviruses (Visser *et al.*, 1993).

From June 1987 until May 1988, hundreds of Atlantic bottlenose dolphins (*Tursiops truncatus*) died along the eastern coast of the United States. Strandings commenced along the coast of New Jersey and eventually spread to the Atlantic coast of Florida. It has been estimated that more than 50% of the inshore population of bottlenose dolphins in this region died (Federal Register, 1993).

An initial investigation concluded that brevetoxin produced by the 'red tide' marine dinoflagellate *Ptychodiscus brevis* was the main cause of the die-off (Geraci, 1989). However, a recent study of tissues from affected animals revealed the presence of morbillivirus infection and associated lesions in more than 50% of animals examined (Lipscomb *et al.*, 1994b).

The most recent known epizootic of morbillivirus disease in cetaceans occurred among bottlenose dolphins in the Gulf of Mexico from June 1993 to mid-1994. This mortality event evolved slowly but eventually affected dolphins from Florida to Texas. The full extent of dolphin mortality in this incident is unknown (Lipscomb *et al.*, 1994a).

Virus isolation or genomic analysis of the morbillivirus present in tissues of bottlenose dolphins from the western Atlantic and Gulf of Mexico epizootics has not been achieved. It is therefore not yet possible to determine the relationships of the morbillivirus or morbilliviruses in these populations to those affecting the harbour porpoise and striped dolphin populations referred to above.

## EPIDEMIOLOGY OF MARINE MAMMAL MORBILLIVIRUSES

Epizootics of morbillivirus infection are believed to have occurred in terrestrial species from antiquity. Measles epidemics in humans date back to the early years of this millenium and waves of rinderpest have raged in large ruminant populations in Africa, Asia and Europe for centuries (Norrby and Oxman, 1990; Scott, 1990). Mortality rates have frequently approached 100%. The epidemiology of these events and our knowledge of experimental morbilliviral infections in animals indicate that these viruses are highly pathogenic agents capable of causing very high mortality in susceptible populations. Since morbilliviral epidemics in terrestrial mammals obviously pre-date the manufacture of organochlorine compounds, it is apparent that these viruses can cause disease outbreaks of epidemic proportions in the absence of contaminants. The pathology of morbilliviral infection in aquatic mammals has been well documented and is very similar to that in terrestrial mammals (Kennedy *et al.*, 1989; Norrby and Oxman, 1990; Domingo *et al.*, 1992; Lipscomb *et al.*, 1994b).

Extrapolating from our knowledge of the epidemiology of morbillivirus infections in terrestrial mammals, morbillivirus epizootics in marine mammals are likely to have resulted from the introduction of morbilliviruses to previously unexposed and therefore immunologically naive populations. Direct contact with an infected animal appears to be the probable method of introduction to a new population. Morbilliviruses frequently cause epizootics in susceptible host species (Norrby and Oxman, 1990; Scott, 1990). They are highly infectious and are excreted in large numbers by many routes including respiratory aerosol, and via a range of body secretions and excretions. Although they are relatively unstable in the environment, their low minimum infectious dose results in a high transmission rate provided the population density exceeds a minimum threshold value. It is therefore not surprising that such viruses can cause major epizootics in marine mammal species.

In non-exposed populations of terrestrial mammals, morbillivirus outbreaks usually affect individuals of all ages, while in those with previous exposure to the virus they predominantly affect young individuals with less developed immune systems (Hoffman, 1983). In the North Sea harbour seal epizootic and the Mediterranean striped dolphin epizootic, mortality centred on adult individuals as well as juveniles. The cause for apparent discrepancy in age-specific mortality in marine mammals between some areas remains unknown, although behavioural factors that would result in a lower exposure of juveniles to the virus have been suggested (Härkönen and Heide-Jørgensen, 1990; Calzada *et al.*, 1994).

## IMMUNOSUPPRESSION IN MORBILLIVIRUS-INFECTED ANIMALS

Lymphoid, epithelial and central nervous system tissues are the major host targets for morbilliviruses. Marked damage to lymphoid tissues has been demonstrated in a wide range of morbillivirus-infected terrestrial and marine mammals including cetaceans (Norrby and Oxman, 1990; Kennedy *et al.*, 1991; Domingo *et al.*, 1992; Duignan *et al.*, 1992; Lipscomb *et al.*, 1994b). The effects of this damage in natural infections are difficult to quantify but clinically significant immunosuppression in morbillivirus-infected marine mammals is

evidenced by the destruction of lymphoid tissues and an increased incidence of secondary fungal, bacterial, protozoal and parasitic infections (Kennedy *et al.*, 1989; 1991; Baker and Raga, 1992; Domingo *et al.*, 1992; Duignan *et al.*, 1992; Lipscomb *et al.*, 1994a; b).

Our understanding of the epidemiology of the aquatic mammal epidemics is incomplete, but the rapid spread of infection and high mortality rates mirror morbilliviral infections in terrestrial animals. These similarities suggest that it is likely that the cause of the recent epizootics in marine mammals was the introduction of a virus into previously unexposed and therefore susceptible populations.

Although there is no evidence that contaminants facilitated recent morbillivirus epizootics in marine mammals, it is well known that organochlorines, including polychlorinated biphenyl compounds (PCBs), accumulate to high concentrations in tissues of marine mammals. These compounds have been demonstrated to reduce immune function in several terrestrial mammalian and avian species (Busbee *et al.*, 1999). Furthermore, altered *in vitro* indices of immune function have been demonstrated experimentally in harbour seals fed fish from environmentally-contaminated waters (De Swart *et al.*, 1994; 1995; Ross *et al.*, 1995) and *in vitro* tests on bottlenose dolphin line cells have shown the capability of organochlorines to induce immune dysfunction in cetacean cells (Busbee *et al.*, 1999). It is therefore likely that such substances could produce immunosuppression in wild marine mammals.

Several studies have attempted to identify a relationship between tissue levels of PCBs and mortality due to morbillivirus infection in marine mammals. For example, higher levels of PCBs were reported in tissues of seals that died during the 1988 European morbillivirus epizootic than in survivors (Hall *et al.*, 1992), whilst exceptionally high concentrations of PCBs were found in tissues of striped dolphins that died during the die-off in the Mediterranean Sea in 1990 compared to concentrations in tissues of this species in years prior to and after this epizootic (Aguilar and Borrell, 1994). Tissue concentrations in bottlenose dolphins that died during the morbillivirus epizootic along the eastern coast of the USA in 1987 and 1988 were also considered to be high although comparisons could not be made with levels in tissues of surviving dolphins (Kuehl *et al.*, 1991). However, under experimental conditions, exposure to PCBs did not increase the susceptibility of seals to morbilliviral disease (Harder *et al.*, 1992).

In essence, these studies provide quantitative data on tissue pollutant concentrations and an indication that some pollutants cause alterations in *in vitro* indices of immune function. However, there is no evidence that they have affected mortality or morbidity due to morbilliviral infection. It should be recognised though, that discovery of a cause and effect relationship between tissue concentrations of contaminants and morbillivirus mortality in these epizootics would be difficult. As morbilliviruses cause direct damage to the immune system and are therefore likely to be a major cause of mortality in infected animals, it is clear that such immunosuppression and any resulting from contaminants will be difficult to quantify separately.

Detection of changes in the prevalence of such diseases can best be achieved by postmortem examination of affected individuals and subsequent correlation of lesions with tissue contaminant concentrations or biomarkers of the toxic effects of these substances. It is currently impossible to separate any possible immunosuppressive effects of contaminants from those due to morbillivirus infection in aquatic mammals that have died as a result of natural morbillivirus infections.

Morbilliviral epidemics are therefore unlikely to be the ideal scenario for investigating possible immunosuppressive effects of contaminants in marine mammals. Such effects are more likely to be insidious and manifest as an increase in susceptibility to neoplasms and diseases caused by organisms normally less pathogenic than morbilliviruses. As in human

immunodeficiency virus infection and in morbilliviral infections in mammals, immunosuppression is associated with an increased incidence of opportunistic fungal, protozoal, bacterial and viral infections. An increase in the rate of neoplasms in a contaminated population, as reported in white whales (*Delphinapterus leucas*) in the St Lawrence Estuary (Martineau *et al.*, 1988), may also be an indication of immunosuppression.

Many morbilliviral-infected bottlenose dolphins and striped dolphins were found in poor body condition (Geraci, 1989; Domingo *et al.*, 1992). Mobilisation of blubber reserves in these animals probably resulted in increased plasma concentrations of organochlorines and a consequently increased risk of toxicity from these compounds. Mortality and morbidity during morbilliviral epizootics may therefore have been higher in dolphins with high body burdens of lipophilic contaminants than in those with lower tissue levels. However, given the lethal effects of morbilliviruses, it is this author's view that it is unlikely that organochlorine toxicity had anything other than a marginal effect on mortality.

In conclusion, the recent epizootics of morbilliviral infection in marine mammals are likely to have resulted from introduction of morbilliviral infection into susceptible populations. The high mortality reported in many of these die-offs is consistent with our knowledge of morbilliviral infections in terrestrial mammals. Although epizootics of infectious disease had not been reported in aquatic mammals prior to 1987 and many of the animals involved in recent die-offs had relatively high tissue concentrations of organochlorines, there is no evidence that contaminants contributed to increased mortality in these populations. However, the increasing data on potentially significant deleterious effects of contaminants on marine mammal health, i.e. immunosuppression, warrants further investigation.

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